Body composition characteristics and body fat distribution in lean women with polycystic ovary syndrome

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Body composition, fat distribution and bone mineral density were examined in lean women suffering from polycystic ovary syndrome (PCOS) and compared with body composition and fat distribution characteristics of weight-matched lean controls. Ten women with PCOS and a body mass index (BMI) below 25.00 (kg/m²) and 10 healthy women with a BMI below 25.00 (kg/m²) matched for age and weight and BMI as controls were enrolled in this study. Body composition and bone density were measured by dual-energy x-ray absorptiometry and fat distribution patterns were calculated. Although matched for age, weight and BMI, lean PCOS patients showed a significantly higher amount of body fat and lower amount of lean body mass than the controls. The majority of PCOS patients showed an intermediate or android kind of fat distribution. Only 30% of the lean PCOS patients corresponded to the definition of gynoid fat distribution while this was true of all lean controls.

Key words: body composition/fat distribution/matched controls/PCOS

Introduction

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting female fertility (Kousta et al., 1999), however its aetiology and pathophysiology are poorly understood. One of the major difficulties of the understanding of the pathogenesis of PCOS is the great variability in symptoms and clinical and biological manifestations of this condition (Franks, 1994; Conway, 1996).

One of the originally described symptoms of PCOS is obesity, which seemed to be associated with anovulation, hirsutism and infertility (Stein and Leventhal, 1935). Therefore the majority of previous studies were performed primarily on obese or overweight women with PCOS. However, obesity is not obligatory in PCOS women and furthermore it cannot be seen as a homogeneous phenotype. Of special importance is the topography of body fat, visible as fat distribution. The major endocrine symptom of PCOS, hyperandrogenicity, is clearly associated with a preponderance of fat localized to upper body sites (Evans et al., 1983). This sex specific fat distribution, commonly called android fat distribution, is associated with obesity and a variety of metabolic characteristics, but is also mentioned as an indicator of reduced reproductive capability of the woman. In this way the phenotype of android fat patterning is in close association with the main symptoms of PCOS, such as being overweight and infertility. Nevertheless, up to now only few studies analysed fat distribution patterns in PCOS patients (Bringer et al., 1993; Douchi et al., 1995; Lefebre et al., 1997). These studies have documented a tendency to centralized or android fat patterning in young women with PCOS. Unfortunately the majority of studies described only fat distribution patterns in overweight PCOS women. The only study analysing body composition and fat distribution pattern in lean PCOS women documented no differences in body composition and fat distribution between lean PCOS patients and healthy controls (Good et al., 1999). These results however, are in contradiction to the evolutionary based assumption that phases of infertility and sterility are associated with android fat distribution and this kind of fat distribution may be an indicator for reduced reproductive capability in a woman. We postulate that even in lean women suffering from PCOS an android type of fat distribution prevails. Therefore the purpose of our study was to analyse body composition, bone density and body fat patterning in lean PCOS women only.

Materials and methods

Subjects

The study was carried out between 1994 and 1997 at the University Clinic for Gynecology and Obstetrics, Department for Endocrinology, Vienna, Austria. The study population consisted of ten Caucasian women aged between 18 and 30 years (mean = 23.9 yrs) with PCOS. All the women suffered from menstrual disorders, such as amenorrhea or oligomenorrhea, and had contacted the Department for Endocrinology because of undesired infertility. The PCOS was diagnosed by ultrasound appearance of polycystic ovaries and determination of hormonal parameters. All women showed hyperandrogenism and the majority of women also showed elevated luteinizing hormone (LH) levels. All probands were classified as normal weight.
with a BMI below 25.00. Ten young women ranging in age from 19–29 years (mean = 22.9) served as controls. The controls were recruited from the staff and students of the University clinic for Gynecology and Obstetrics and were matched for age and weight status of the PCOS group. All controls had regular menstrual cycles (26–33 day cycles) and age specific normal sex hormone levels. All probands, PCOS patients as well as controls, were in good health and were not on any medication which might affect hormone metabolism or body composition, even hormonal contraceptives were stopped for a minimum of four months prior to the present investigation. All subjects were non-smokers and none of them was on excessive physical training. There was no population bias between patients and controls in the present study: both were of the same ethnic origin, and PCOS patients as well as controls stemmed exclusively from Vienna or the neighbouring Lower Austria. All probands gave their written informed consent.

**Hormone concentrations**

The examination started with the quantitative determination of 17β-oestradiol, follicle stimulating hormone (FSH), LH, testosterone, dehydroepiandrosterensulphate (DHEA-S), androstendione and sex hormone-binding globulin (SHBG). Blood samples were collected between 7.30 and 9.30am before day 10 of the cycle. The quantitative determination was made at the central hormone laboratory of the University Clinic for Gynecology and Obstetrics.

**Anthropometrics**

Stature (in cm) and body weight (in kg) was determined for each proband according to previously published methods (Knussmann, 1988). For a better description of the weight status the BMI was calculated as: weight in kg divided by the square of height in metres. Weight status was classified using the following BMI categories according to the World Health Organization (WHO, 1995):

- **Thinness:** grade 1 BMI 17.00–18.49 (mild thinness)
- grade 2 BMI 16.00–16.99 (moderate thinness)
- grade 3 BMI < 16.00 (severe thinness)

- **Normal range:** BMI 18.50–24.99

- **Overweight:** grade 1 BMI 25.00–29.99 (mild overweight)
- grade 2 BMI 30.00–39.99 (severe overweight)
- grade 3 BMI > 40.00 (obese)

**Body composition**

Body composition analyses were performed before day 10 of the cycle. Dual-energy x-ray absorptiometry (DEXA) (Hologic 2000) was used to measure bone, lean and fat mass (Blake and Fogelman, 1997). Although this method is indirect, its high reliability, relatively low costs and comfort for the probands make the dual energy x-ray absorptiometry especially useful for the determination of body composition. By DEXA, the body consists of soft tissue, i.e. fat and lean tissue and bone. DEXA measures total body bone mineral content (BMC) and density, fat mass and lean mass with a precision of 0.9, 4.7 and 1.5% respectively. The precision for the abdominal fat mass and fat percentage is 4.3 and 3.4% respectively. The extinction of x-rays, which is dependent on the tissue, is measured and absolute and relative fat mass and lean body mass are estimated. The scanner uses an x-ray source, an internal wheel to calibrate the bone mineral content and an external luciate and aluminium phantom to determine the percentage of fat of each soft-tissue sample scanned. Simultaneous with the measurement of the skeleton, the percentage of fat is determined from the ratio of attenuation of the lower energy (70kVp) to that of the higher energy (140kVp) of the beam. This is calculated for all non-skeleton pixels scanned and extrapolated over the skeleton-containing pixels. The relatively low radiation dose with 0.1m Sievert and a short scanning time (<7 min) make this technique especially suitable for such determinations. Scanning was done by the Hologic total body scanner. A phantom, especially constructed for body composition determination and calibrated for fat and lean mass and bone mineral content, was placed beside the proband. Default software readings provided lines positioned to divide the body into six compartments, i.e. head, trunk, arms and legs. The trunk was defined by a horizontal line below the chin, vertical lines between trunk and arms and a lower border formed by oblique lines passing through colli femuri. The region below this lower border of the trunk, including both legs and the hip region is called lower body region. For each region of the whole body fat and lean body mass and BMC were determined.

**Fat distribution**

For a better description of the sex specific fat distribution the fat distribution index (FDI) (Kirchengast et al., 1997a) was calculated:

\[
FDI = \frac{\text{Upper body fat mass in kg}}{\text{Lower body fat mass in kg}}
\]

A fat distribution index below 0.9 indicates a gynoid fat distribution, i.e. the fat mass of the lower body surpassed the fat mass of the upper body. A fat distribution index >1.1 defines an android fat distribution. In this case the amount of fat tissue of the abdominal region surpassed the fat mass of the lower body. An FDI between 0.9 and 1.1 is classified as an intermediate stage of fat distribution. We used the FDI for quantification of the fat distribution instead of the widely used waist to hip ratio, because the waist to hip ratio describes body shape and silhouette but not the quantitative amount of fat distribution. Nevertheless we have to be aware that the FDI describes not the ratio of abdominal fat to gluteal-femoral fat, but the ratio between upper body fat, including abdominal fat and breast fat mass, and lower body fat.

**Statistical analysis**

The statistical analyses were carried out using SPSS Version 7.0 (Microsoft Corp.) according to a previously published method (Bühl and Zötfel, 1996). After computing descriptive statistics (means, SD) group differences were tested regarding their significance using paired student t-tests. Since the results of the Kolmogorov-Smirnov test indicated that no normal distribution could be assumed for the hormonal variables, for statistical analysis of group differences in hormone levels the non-parametric Wilcoxon test for paired samples was applied. Furthermore linear regression analysis was computed in order to test the impact of body mass and body composition on fat distribution patterns.

**Results**

**Body size, body composition and hormone levels**

As to be expected, the two proband groups differed in stature, body weight and BMI only insignificantly. In contrast, regarding body composition parameters, significant group differences were observed. Although matched for weight and weight status, lean PCOS patients and lean controls differed significantly in absolute and relative amount of body fat. PCOS patients show a significantly higher amount of fat tissue of the total body and the upper body region, while no significant difference of the lower body fat mass was observable between the two proband groups. Regarding lean tissue mass, significantly higher values were found for the controls, this was true of the lean mass of the total body as well as of the upper
Fat distribution of women with PCO syndrome

Table I. Body size, fat distribution and body composition variables in lean PCOS patients and lean controls; paired Student t-tests

<table>
<thead>
<tr>
<th></th>
<th>PCOS lean</th>
<th>Lean controls</th>
<th>t-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Stature (cm)</td>
<td>167.2</td>
<td>5.1</td>
<td>167.6</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>57.3</td>
<td>3.8</td>
<td>57.2</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>20.7</td>
<td>7.4</td>
<td>20.4</td>
</tr>
<tr>
<td>Fat distribution index</td>
<td>1.01</td>
<td>0.23</td>
<td>0.57</td>
</tr>
<tr>
<td>Total fat mass (kg)</td>
<td>21.2</td>
<td>5.4</td>
<td>14.8</td>
</tr>
<tr>
<td>Upper body fat mass (kg)</td>
<td>9.1</td>
<td>3.3</td>
<td>4.5</td>
</tr>
<tr>
<td>Lower body fat mass (kg)</td>
<td>8.8</td>
<td>1.8</td>
<td>7.9</td>
</tr>
<tr>
<td>Total fat %</td>
<td>35.7</td>
<td>7.6</td>
<td>26.4</td>
</tr>
<tr>
<td>Total lean mass (kg)</td>
<td>35.6</td>
<td>3.9</td>
<td>38.7</td>
</tr>
<tr>
<td>Upper body lean mass (kg)</td>
<td>18.7</td>
<td>2.5</td>
<td>20.8</td>
</tr>
<tr>
<td>Lower body lean mass (kg)</td>
<td>11.1</td>
<td>1.3</td>
<td>11.9</td>
</tr>
<tr>
<td>Total bone mass (g)</td>
<td>2065.7</td>
<td>276.7</td>
<td>2312.7</td>
</tr>
<tr>
<td>Bone density (g/cm²)</td>
<td>1.080</td>
<td>0.094</td>
<td>1.142</td>
</tr>
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</table>

Table II. Sex hormone concentrations in lean PCOS patients and lean controls. Wilcoxon test for paired sample

<table>
<thead>
<tr>
<th></th>
<th>PCOS lean</th>
<th>Lean controls</th>
<th>z-value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>FSH (mIU/ml)</td>
<td>5.36</td>
<td>2.81</td>
<td>5.57</td>
</tr>
<tr>
<td>LH (mIU/ml)</td>
<td>5.4</td>
<td>2.5</td>
<td>4.05</td>
</tr>
<tr>
<td>Oestradiol (pg/ml)</td>
<td>34.7</td>
<td>15.6</td>
<td>51.3</td>
</tr>
<tr>
<td>Testosterone (ng/ml)</td>
<td>0.73</td>
<td>0.21</td>
<td>0.31</td>
</tr>
<tr>
<td>Androstendione (ng/ml)</td>
<td>3.39</td>
<td>1.55</td>
<td>1.67</td>
</tr>
<tr>
<td>DHEA-S (ug/ml)</td>
<td>2.47</td>
<td>0.67</td>
<td>1.39</td>
</tr>
<tr>
<td>SHBG (nmol/l)</td>
<td>48.2</td>
<td>19.4</td>
<td>54.5</td>
</tr>
</tbody>
</table>

DHEA-S = dehydroepiandrosterone sulphate
SHBG = sex hormone-binding globulin

body. No significant differences between the two proband groups occurred in lower body lean mass.

Lean PCOS patients exhibited the significantly lower values in absolute bone mineral content, while the group differences of bone density of the total body were not of statistical significance (Table I).

As to be expected PCOS patients exhibited higher LH and androgen levels. Nevertheless only the group differences of testosterone and DHEA-S were of statistical significance (Table II).

Fat distribution and fat-lean ratios

The fat distribution patterns of only 30% of the lean PCO patients were classified as gynoid with a fat distribution index below 0.9. 50% corresponded to the definitions of android fat distribution (FDI >1.1) and 20% represented an intermediate stage of fat distribution (FDI between 0.9 and 1.1). The threshold to discriminate between gynoid, android and intermediate fat patterning are in accordance with published definitions (Kirchengast et al., 1997a). The lean controls exhibited exclusively a gynoid type of fat distribution (Table III). Therefore significant differences of the FDI occurred. The FDI of the PCOS patients was significantly higher (P ≤ 0.000) than the FDI of the lean controls (Table I). The linear regression analyses analysed the impact of weight status, and absolute and relative amount of body fat on fat distribution for each proband group separately. A significant association between the fat distribution and the body composition parameters mentioned above was only found for the lean PCOS patients. In this group the FDI increased with increasing absolute and relative fat mass. In case of relative fat mass this was also true of lean controls (Table IV and Figure 1 A,B,C).

Discussion

Signs of potential reproductive success are classified as attractive. Cross-cultural studies show that moderate plumpness and a gynoid type of fat patterning are considered as typical signs of female attractiveness in the majority of investigated cultures (Brown and Konner 1987; Brown 1991; Anderson et al., 1992; Singh 1993, 1994; Singh and Luis, 1995). This association between body fat mass and fat distribution and attractiveness may be due to the importance of body fat and gynoid fat patterning for potential reproductive success. A sufficient amount of body fat is absolutely necessary for the onset and maintenance of regular and ovulatory menstrual
Table IV. Linear regressions analyses

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>SE of B</th>
<th>Beta</th>
<th>T</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PCOS lean</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>0.04</td>
<td>0.06</td>
<td>0.27</td>
<td>0.79</td>
</tr>
<tr>
<td>total fat mass</td>
<td>0.03</td>
<td>0.01</td>
<td>0.78</td>
<td>3.58</td>
</tr>
<tr>
<td>fat %</td>
<td>0.02</td>
<td>0.01</td>
<td>0.73</td>
<td>3.02</td>
</tr>
<tr>
<td><strong>lean controls</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI</td>
<td>0.03</td>
<td>0.01</td>
<td>0.49</td>
<td>1.58</td>
</tr>
<tr>
<td>total fat mass</td>
<td>0.02</td>
<td>0.01</td>
<td>0.44</td>
<td>1.39</td>
</tr>
<tr>
<td>fat %</td>
<td>0.02</td>
<td>0.01</td>
<td>0.56</td>
<td>1.99</td>
</tr>
</tbody>
</table>

BMI = body mass index.

of the hormonal situation and reproductive status of a woman. Peripheral fat tissue, especially in the lower body region is an important source of extra-ovarian oestrogen synthesis, because the aromatization from androgens to oestrogens takes place there. The gynoid type of fat distribution develops during female puberty and persists during the fertile phase of adult life and remains stable even under worse conditions such as malnutrition and phases of amenorrhoea caused by undernutrition (DeRidder et al., 1990, Frisch, 1990). At the end of the fertile phase the gynoid type of fat distribution changes through an intermediate stage when the amount of upper and lower body fat are more or less equal to the android type of fat distribution typical for the post-menopause when reproductive capability has ended irreversibly (Ley et al., 1992; Kirchengast et al., 1997b). A post-menopause android fat distribution pattern is seen in association with severe over weight and obesity during fertile phase, and especially in women suffering from metabolic diseases and/or polycystic ovaries (Björntorp 1988, 1997; Lefebvre et al., 1997; Kirchengast et al., 1998). However PCOS is often associated with overweight or obesity and the android fat distribution patterns may be the result of being overweight only. This assumption is supported by the finding of no differences in body fat distribution between lean PCOS patients and lean controls (Good et al., 1999). In contrast in our present study we found significant differences in body composition and fat distribution between lean women with PCOS and lean controls. Although lean PCOS patients and lean controls were matched for weight, lean PCOS women had a significantly higher amount of body fat, and a significantly lower amount of lean body mass than the lean controls. Furthermore, significant differences occurred in bone mass: lean PCOS women showed a significantly lower total bone mass and an insignificantly lower bone density than the lean controls. This finding is in contradiction to the well described positive effect of androgen excess on bone density (Dixon et al., 1989; DiCarlo et al., 1992; Simberg et al., 1996; Dagogo-Jack et al., 1997). However, the majority of studies were performed on overweight or obese PCOS patients only. The observed higher bone density and bone mineral content in overweight or obese PCOS women may be explained by the assumption that obesity or overweight may increase bone mineral density through biomechanical forces (Dumesic et al., 1998; Ferretti et al., 1998) or by causing increased

cycles. Even during adult life a marked decrease of adipose tissue may result in secondary amenorrhoea. Body fat stores in females, especially at the lower body region (Rebuffe-Scrive et al., 1985, 1986, 1990) were interpreted as energy stores for times of increased physiological stress such as pregnancy or lactation but also as energy deposits for phases of malnutrition and food shortages – a situation not uncommon during our evolution and history and even today in many parts of the world. On the other hand the amount of body fat and especially the sex specific kind of fat distribution are indicators
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sterility was consequently historically extraordinarily rare. In contrast, PCOS is the most common endocrine disorder affecting fertility during adult life and is not strongly associated with severe obesity. In our sample we found an extremely high prevalence of android or intermediate fat distribution in PCOS women, even in lean ones. There is a cross-cultural association of female unattractiveness in relation to the body shape resulting from the android or intermediate fat distribution pattern. This could be seen as a consequence of the typical body shape of PCOS affected women.

Acknowledgements

The authors are gratefully indebted to their probands without whose co-operation the present study could not have been performed.

References


Aromatization of androgens to oestrogens (Kley et al., 1980; Lobo et al., 1981). The low BMD of the lean PCOS patients is also in marked contrast to the results of Good et al. (1999) who reported increased BMD for lean PCOS patients in comparison to healthy controls. However Good et al. (1999) documented a significant higher bone density for the upper extremities and the left ribs only and regarding total body bone density no differences between patients and controls were reported (Good et al., 1999).

Beside body composition and bone density the lean PCOS patients and lean controls differed significantly in their body fat distribution, however we have to state that body fat distribution was determined using the FDI only and not the widely used waist hip ratio (WHR). In our opinion, the FDI, which describes the quantitative ratio of upper to lower body fat, is an adequate measure of the fat distribution and may determine fat distribution to some extent better than the WHR, which describes first of all the body silhouette. We are aware that the amount of upper body fat does not only describe the amount of abdominal fat, but also the fat mass of the breasts, however, the fat mass of the breasts increases with increasing body weight and obesity but the PCOS patients of our sample were lean, normal weight and did not differ in weight status from the healthy controls. Furthermore we found no paper describing an increased breast size and an increased fat mass of the breasts in PCOS patients. Therefore we assume that the inclusion of breast fat into FDI plays no significant role in our findings. Good et al. (1999) used the upper body to lower body fat ratio too, beside the WHR (Good et al., 1999). Nevertheless our results differed from that of Good et al., (1999). While in our sample healthy controls exhibited gynoid fat patterning exclusively, the majority of lean PCOS patients (70%) showed a non gynoid type of fat patterning and the fat distribution patterns of 50% of the lean PCOS patients were classified as android. Good et al. (1999) found no differences in fat distribution between PCOS and lean controls, this was true of WHR as well as upper body to lower body fat ratio. These differences between our results and those of Good et al., (1999) should not be over interpreted because sample size in both studies was small. Furthermore Good et al. (1999) used a BMI <26.00 as the threshold value to exclude overweight probands from their sample. In the present study we used a BMI <25.00 as the threshold value according to the definitions of the World Health Organization (WHO, 1997). Therefore, the mean BMI of the probands was higher in the study of Good et al. (1999) than in our sample (22.4 versus 20.7). So the differences in the results of Good et al., (1999) and the present paper may be due to the different mean weight status (BMI) of the two samples.

In our sample PCOS affected women showed an extraordinarily high prevalence of android or intermediate fat distribution, even lean women. Therefore the majority of PCOS affected women did not correspond to the cross-cultural constant standard of attractive female body shape. During our evolution and history only a few women were excessively obese during their fertile years or reached the post-menopause. The observation that android fat distribution in association with obesity or menopause is an indicator for reduced fertility or irreversible


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